UNCINARIOSIS (ANKYLOSTOMIASIS). A FURTHER REPORT OF A CASE, WITH NOTES UPON THE AUTOPSY.[1]

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In view of the fact that a number of cases of uncinariosis have been discovered in the United States (Missouri, New York, Louisiana, District of Columbia, Texas, Virginia, Maryland—not all yet reported) in the last few years, the majority in the past few months, and since, to many of us, unless engaged in teaching, it is little more than a name, it seems advisable to bring this serious disease to the attention of the profession in some detail.

It is my belief, from the nature of the disease, as well as from recent developments, that it is far more common among native Americans than has been the general impression heretofore. Besides, there is the ever-increasing danger of its importation in emigrants from localities where the disease is prevalent, and in those returning from our newly acquired possessions.

Uncinariosis (ankylostomiasis) is a chronic disease due to infection by a small thread-worm known as the uncinaria duodenalis (ankylostoma duodenalis), which is characterized clinically by intense anæmia, dyspnœa, weakness, gastro-intestinal disturbances, and, in advanced cases, by œdema of the face and extremities and serous effusions into the cavities of the body.

History. The disease has been described under various names: tropical chlorosis, Egyptian chlorosis, geophagia, mal d'estomach, cachexie aqueuse, cachexia africana, tunnel disease, miner's disease, etc.

In the *Papyros Ebers* (German translation by Dr. H. Joachim), the oldest book upon the subject of medicine, written in the middle of the sixteenth century before Christ, is recorded a disease among the Egyptians known as the ā a ā, which so strongly resembles uncinariosis in its symptomatology that it seems at least possible that they are identical. The similarity is further strengthened by the mention of remedies given for worms in the intestines of those afflicted with this disorder.

From the fact that it is several times spoken of as the deadly ā a ā, it must have been very fatal in those ancient times.

According to Sandwith[2] the first mention of the disease in modern times was in Brazil in 1648, and in the West Indies and in

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[1] Read before the Medical Society of the District of Columbia, November 6, 1901.

[2] Observations on Four Hundred Cases of Ankylostomiasis, Lancet, 1894, vol. I.

Guiana in the early part of the eighteenth century, while in Europe it was first noted among the miners of Auzin in 1802.

To Angelo Dubini, of Milan, is due the credit of discovering the nematode in man which is the cause of the disease. He, in 1838 (published 1843), found the parasite[1] in the small intestine of a woman dead of pneumonia; but the importance of the discovery was not realized until 1854, when W. Griesinger declared, upon the evidence of a single autopsy, performed in Cairo, April 17, 1852, that Egyptian chlorosis was caused by the ankylostoma duodenalis.[2] This fact was apparently lost sight of until, in 1866, it was confirmed by Wucherer.[3]

The disease came into prominence in Europe in 1879 or 1880, when a number of workmen in the St. Gothard tunnel developed a severe form of anæmia, which was found to be uncinariosis.

The geographical distribution is wide, the disease having been reported from most of the countries of Europe, in Africa (especially Egypt), Australia, South America, the Indies, the United States, and other localities. As would be expected, it is more common in the tropical and subtropical countries. Sandwith states that the parasite has been found between the parallels of 52° N. and 30° S., but that above 47° N. it can develop only in sheltered localities, such as mines, tunnels, etc.

As our possessions, excepting Alaska, lie entirely below the 50th parallel, there seems to be no reason why we should not meet with uncinariosis more frequently than we have heretofore.

I am told by Dr. Stiles that parasites of the same genus as that found in man cause the death of many dogs in this locality, and are very common in Alaska in the fur-bearing seal, and through the South in sheep, goats, and cattle.

There is thus far no proof, however, that these parasites are transmissible to man.

According to Manson, in Egypt the parasite is found in nearly every cadaver, and in India it was recovered from 75.58 per cent. of 1249 natives to whom thymol had been administered.

It must not be supposed, however, that all those who are infected show the symptoms of uncinariosis; far from it, many are strong and healthy. The weak and poorly nourished are those who are least able to withstand the loss of blood occasioned by the parasite.

PARASITE. The worm is nearly cylindrical in shape, from 0.4 to 1 mm. ($\frac{1}{60}$ to $\frac{1}{25}$ inch) in diameter, and from 6 to 18 mm. ($\frac{1}{4}$ to $\frac{3}{4}$ inch) in length. It is yellowish or grayish-white in color, with translucent edges. The males are more slender and more transparent than the females by reason of the less extensively developed sexual organs. The head end is always bent backward. The tail, which can be distinguished by the swelling at the end (the bursa copulatrix) is much more curved than the head, sometimes appearing even to be rolled up. The length of the mature male is from 6 to 11.5 mm. The diameter is nearly the same for the posterior two-thirds (about 0.5 mm.), but the anterior third tapers down and ends in a truncated cone at the head. The female is usually larger (from 6 to 15 mm.), much thicker (about 1 mm.), and is generally found slightly curved dorsally. The number of the females is, as a rule, much larger than that of the males.[1] The armamentarium of the parasite consists in four claw-like hooks set in the margin of the mouth capsule, two on either side of the ventral line, and two conical teeth, one on each side of the dorsal line. By means of these the worm clings to the mucous membrane of the small intestine and sucks the blood from its host.

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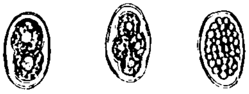
[1] Dubini gave the name of ankylostoma duodenalis to the worm, which, according to zoölogists, is wrong, since the genus is identical with that found in the wolf, in 1789, by Frölich, and called by him uncinaria.

[2] Archiv für Physiologische Heilkunde, 1853-1854.

[3] Gazeta Medica da Bahia, 1866, and Archiv f. klin. Med., 1872.

From the fact that the blood cells are found unchanged in the parasite, it is believed that only the serum is used for its nourishment; thus it becomes a most wasteful feeder.

The ova, which are a perfect oval in shape, measure 0.055 to 0.065 mm. by 0.032 to 0.043 mm., and are covered with a transparent shell through which may be seen the segmented yolk.



Ova of the uncinaria duodenalis (multiplied about 300 times).

Looss[2] has proved by experiment that the development of the egg depends upon the surrounding temperature, its accessibility to the air, the amount of moisture, and upon the character of the culture media. Thus he found that in Alexandria, Egypt, where the temperature in summer varies around 80° F., the eggs on the surface of the media develop and the embryos escape in twenty-four hours. If a cool night intervenes, with a temperature of from 70° F. to 72° F., these changes do not occur for thirty-six to forty hours, and that a temperature of 33.8° F. kills the eggs in twenty-four to forty-eight hours. He also found that too much moisture not only delayed the development of the eggs, but caused the death of a large number of them.

About the second or third day the rhabditiform embryo, when under favorable circumstances, undergoes the first ecdysis—*i. e.*, casts its cuticle, which has by this time become loosened. From the fifth day on the second ecdysis takes place. The cuticle is seen to divide into two layers, which separate more and more until the outer cuticle is separated from the body, but remaining around it, thus giving rise to the theory of encystment.

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[1] J. Ch. Huber. Twentieth Century Practice of Medicine.

[2] Centralbl. f. Bakter. u. Parasitenkunde, 1896.

The young worm has now reached the infection stage and is ready to enter its host and begin its parasitic life. Looss has succeeded in keeping worms alive in this condition for three months. The ripe embryo prefers water as its medium, and may thus easily be taken into the system. He has shown that both eggs and embryos are killed by being dried out, which tends to disprove the theory of infection through dust which may be blown about.

Looss[1] estimates that about four or five weeks are necessary after infection for the worm to become fully mature, and that in Egypt five or six weeks is the probable period necessary for the freshly laid eggs to develop into individuals containing eggs.

As there is no development of the eggs in the host it follows that any new infection must come from outside. This seems a very fortunate circumstance, as unless the primary infection is a large one, or the exposure is repeated, there will be no ill effects in the majority of instances.

The parasites, which sometimes number thousands, are said to be found in greatest abundance in the jejunum, secondly in the duodenum, and to a lesser extent in the ileum. This, however, was not my experience (see autopsy). The young worms change their positions frequently, thus allowing a large amount of blood to escape from the wounds deserted by them for new fields.

MODE OF INFECTION. The embryo finds its most favorable habitat in damp soil, hence the vast majority of those infected are either earth eaters or are engaged in work which brings their hands in contact with the earth. Thus miners, gardeners, workers in brickyards, etc., are especially subject to the disease. Sandwith found in 200 cases carefully studied that 190 worked in more or less damp soil with their hands. These people probably convey the infection to their mouths by eating without having previously cleansed their hands. Some, no doubt, are infected through drinking-water, but Giles' experience does not bear out this theory. He examined fifty-six water-supplies of infected villages, and found one doubtful embryo.

F. M. Sandwith[2] rather supports the idea suggested by Looss, that infection may take place by the penetration of the embryo through the skin, and quotes an experience of that author as follows: Looss accidentally let a drop of water which contained numerous embryos fall upon his hand. Noticing that the spot became red and burned, he let fall another drop, which produced the same result. On careful examination of the area he found countless sheaths which had been discarded by the embryos, indicating that they had thrown off their outer covering and entered the skin. Shortly thereafter he developed symptoms of uncinariosis and found ova in his evacuations. This, however, was not positive proof that he was infected at this time, as he had previously suffered from the disease. Looss then tried several experiments to prove that the embryos could penetrate the skin, one of which was successful. He allowed a drop of infected water to fall on the leg of a boy who was to have the limb immediately amputated. That portion of skin was then hardened, and sections made which showed that the embryos had entered the skin by way of the hair follicles.

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[1] Centralbl. f. Bakter. u. Parasitenkunde, 1897, p. 913.

[2] British Medical Journal, September 14, 1901.

Whether the embryos are able, after penetrating the skin, to reach the intestinal tract remains to be proved.

MORBID ANATOMY. "The pathological anatomy is that of extreme anæmia plus the local lesions in the small intestine due to the presence of the uncinaria." There is general bloodlessness of all the tissues; œdema and effusions into the cavities; the fat is usually fairly well preserved; the heart is enlarged, dilated, and may have undergone fatty change; the spleen and kidneys are very often lardaceous and waxy, or the spleen, and still more frequently the liver, presents a certain degree of atrophy. If the autopsy is performed a short time after death the parasites may be seen clinging by their mouths to the mucous membrane of the small intestines. If the body has grown cold they are found in the mucus.

The intestinal wall may be thickened from a catarrhal process. Scattered about the intestine in and under the mucous layer, and visible through the serous and muscular coats, may be seen small ecchymotic spots which, on close inspection, show minute punctures indicating the points where the parasites are attached.

In a few instances parasites have been found in small cavities filled with blood under the mucous membrane. The cavities communicate with the interior of the intestine by small punctures through which the worms have gained an entrance. The intestinal contents may be streaked with blood, and very occasionally there may be extensive hemorrhage.

The intense anæmia is probably due, first, to the actual abstraction and leakage of blood from the mucous membrane of the intestine; secondly, to interference with the proper digestion and absorption of food because of a catarrhal process set up in the intestine by the bites of the parasite; and, lastly, it has been suggested that there is an absorption of poisons produced by the parasite itself or from the intestinal contents through the wounds made in the mucous membrane which aids in causing the anæmia.

There is apparently no actual hæmolysis, as the amount of iron in the liver is not increased, as is the case in pernicious anæmia.

SYMPTOMS. According to Manson[1] "the essential symptoms are those of a progressive anæmia, an anæmia which is usually associated with dyspeptic trouble, but which, in uncomplicated cases, is not associated with wasting. If the progress of the case is unchecked serous effusions in different organs and fatty degeneration of the heart ensue, and death may occur from syncope or intercurrent complications." There may be either diarrhœa or constipation, or the bowels may be regular. Occasionally the evacuations have a reddish-brown shade, due to the admixture of blood, and very rarely there may be distinct hemorrhage from the intestine. The appetite is variable; it may be excessive, capricious, or lost entirely. In advanced cases there is dyspnœa on slight exertion, with palpitation, œdematous swelling of the face, feet, and ankles, and extreme weakness. The urine is usually alkaline or neutral, with a specific gravity of 1010 to 1015; a trace of albumin may be noted in advanced cases. There is often stupidity so dense as to amount, in some cases, almost to weak-mindedness. If a severe infection occurs before puberty that period is apt to be delayed. The temperature is said, as a rule, to be subnormal, though there may be transient flashes of fever. The heart is usually enlarged, and hæmic murmurs are to be heard at the base and over the vessels of the neck. As a rule, there is no enlargement of either liver or spleen. The blood shows the following changes: A marked reduction in the red cells, sometimes below 1,000,000; a proportionate reduction in the hæmoglobin, poikilocytes, and nucleated red cells. Some observers have noted megaloblasts as well as normoblasts.

Of nineteen cases studied in Porto Rico by B. K. Ashford,[2] fourteen showed normoblasts and seven megaloblasts. The megaloblasts were, however, never in excess of the normoblasts (an excess of megaloblasts is considered quite characteristic of pernicious anæmia).

There is no leucocytosis in uncomplicated cases. Ashford noted an excess of eosinophiles in several cases.

DIAGNOSIS. It has very aptly been said that the way to make a diagnosis of uncinariosis is "first to suspect its presence." Once having had the suspicion aroused by the peculiar anxious expression of the face and the intensely anæmic appearance of the patient, it is a very simple matter to examine the stools for the ova, which are present usually in large numbers. The parasites themselves are almost never seen until after the administration of an anthelmintic.

Both ova and parasites are readily recognized from the cuts in works on clinical diagnosis.

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[1] Tropical Diseases.

[2] New York Medical Journal, 1900.

If a microscopist is not at hand, the administration of thymol to suspects would cause the parasites to appear in the stools, where they may be seen with the naked eye.

The disease bearing the closest resemblance to uncinariosis is pernicious anæmia. The similarity is most striking in the insidious onset, pallor, gastro-intestinal disturbances, circulatory symptoms, weakness, dyspnœa, œdema, tendency to periods of improvement followed by relapses, marked reduction in the number of erythrocytes, presence of poikilocytosis and nucleated red cells, and absence of leucocytosis.

It is said that in uncinariosis the color index is usually low, while in pernicious anæmia it is high—*i. e.*, there is a greater proportional reduction in hæmoglobin in uncinariosis; but this is not always the case. In fact, it is many times almost impossible to differentiate between the two diseases without an examination of the evacuations to determine the presence or absence of the ova.

In leukæmia the blood is very different, as a rule, though similarity has been reported.

True chlorosis is readily differentiated by examinations of the blood and feces; malarial cachexia by the enlarged spleen, presence of plasmodia and absence of the ova of the uncinaria.

In every case of anæmia not attributable to some definite cause the evacuations should be examined microscopically for the ova of the uncinaria.

PROGNOSIS. If the diagnosis is made and proper treatment instituted before the disease has existed long enough to produce serious changes in the tissues and organs of the victim, recovery is prompt; but if the condition goes unrecognized for months or years the result is not infrequently fatal. A small number of cases are very acute and severe, death resulting in a few weeks after the first appearance of symptoms. As a rule, however, the course extends over some years.

Of Sandwith's 400 cases, 89.5 per cent. were cured, or greatly improved; 2.5 per cent. were unimproved, and 8 per cent. died

In prolonged cases there are usually periods of temporary improvement.

Even if death does not result directly from uncinariosis, those afflicted are especially prone to succumb to some intercurrent disease.

TREATMENT. The recognized treatment is thymol administered as follows: 15 to 30 grains repeated four times at intervals of one and one-half to two hours. If the bowels do not act spontaneously in twelve hours after the last dose, give a purgative, which is usually not necessary. It is well to clear the bowels the day before with a purgative, and place the patient on liquid diet. The fact that thymol is poisonous must not be lost sight of, and the patient should be carefully watched. Since the drug is freely soluble in alcohol, ether, turpentine, chloroform, oil, glycerin, and certain alkaline solutions, these should be withheld during its administration.

In seven or eight days the stools should again be examined, and if the ova are still present the same programme should be carried out and repeated until all the parasites have been destroyed.

The treatment of the anæmia from uncinariosis is the same as in that secondary to any other cause.

History in the United States. It seems very probable that the form of anæmia spoken of by Jean Louis Chabert (1821), James B. Duncan (1850), and others, as existing to a considerable extent in our Southern States was uncinariosis.

The number of authentic cases, however (in which the diagnosis was established by the finding of the ova), reported by observers in the United States is as yet small, and, as far as I have been able to learn, appeared in the following order:

The first by W. L. Blickhahn,[1] of St. Louis (1893); five cases by F. G. Möhlau[2] (1896); one case by C. H. Tebault, Jr.[3] (1899); one by J. H. Dyer[4] (March, 1901); my own case[5] (June, 1901); Allyn's case[6] (July, 1901); one case by Wm. B. Gray[7] (September, 1901); and one by M. Charlotte Schaefer,[8] from Texas (October 26, 1901), making twelve in all. Of these twelve cases, five undoubtedly had their origin in the United States; three probably brought the infection from abroad, one from Mexico having been in the United States only a short time when symptoms appeared; in three the data upon which to base an opinion upon this point was not given.

Dr. Gray also reports a second case, a sister of the one noted above, but as neither ova nor parasites were found it does not seem justifiable to include it among the authentic cases.

My friend Dr. Allen J. Smith, of the University of Texas, writes me that he is inclined to believe that the disease is quite common in Texas, since he found the ova in the evacuations of eight out of eighty-eight students. The majority of these cases, Dr. Smith says, have never been outside of the State. His forthcoming paper upon his observations will be very interesting.

G. R., American, aged nineteen years, of Westmoreland County, Va., occupation, light farm work, was admitted to the Garfield Hospital, of Washington, D. C., May 31, 1901. The patient was a small baby at birth, but was said to have developed normally until about three years previous to his admission. This, however, was probably a mistake on the part of his friends, as the boy's development was that of a lad under fourteen. He was small, childish in voice and manner, and the genital organs were infantile. His mentality was much below the average; in fact, he seemed very stupid and dull. He had never had any of the diseases of childhood, nor had he had malaria. About three years prior to his admission it was noticed that the boy was pale; one year later the anæmia became very marked, and there was swelling of the face, abdomen, and feet. There was, however, an improvement until about three months before he came to the hospital.

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[1] Medical News, December 9, 1893.
[2] Buffalo Medical Journal, 1896-1897.
[3] New Orleans Medical and Surgical Journal, 1899-1900.
[4] Interstate Medical Journal, St. Louis, March, 1901.
[5] Philadelphia Medical Journal, June 29, 1901.
[6] American Medicine, July 13, 1901.
[7] Virginia Medical Semi-Monthly, September 27, 1901.
[8] Medical News, October 26, 1901.

The chief symptoms were great weakness, dyspnœa on the slightest exertion, and pallor. Contrary to the rule in these cases, there was no complaint of any gastro-intestinal disturbance.

The expression was anxious and dejected. The skin was of a faint yellow tint; the face was bloated, especially about the eyes.

The mucous membranes were extremely pale, the body well nourished, but the muscles flabby. The tongue was clean. The heart's impulse was widely diffused, the apex-beat was in the fifth interspace and almost as far to the left as the mid-axillary line. Over the base there was a loud systolic murmur, the point of maximum intensity being in the pulmonary area. This murmur was also heard in the vessels of the neck. The second pulmonary sound was accentuated. At the apex was heard a systolic murmur which was transmitted to the axilla and to the angle of the left scapula. A faint diastolic murmur was also present over a small area near the apex. The feet and ankles were œdematous. The urine was clear, pale, acid, and showed a specific gravity of 1010. The lowest blood count, June 4th, showed the red cells to be 1,450,000 per cubic millimetre (29 per cent. of normal), the whites 4400 (slightly below the average), hæmoglobin 30 per cent. Dr. J. B. Nichols' report upon the stained cover-slips, June 9th, is as follows: Macrocytes and microcytes a few; polychromatophilia, not observed; poikilocytes, a few; microblasts, normoblasts, and megaloblasts, none.

Leucocytes: Small mononuclear, 15 per cent.; large transitional, 9 per cent.; polynuclear, 71 per cent.; eosinophiles, 5 per cent.; microscopical examination of the evacuations showed innumerable ova of the uncinaria, upon which the diagnosis of uncinariosis was made, and later confirmed by Dr. C. W. Stiles, of the Department of Agriculture, who very kindly saw the case with me and gave valuable advice as to the recovery of the parasites, etc. No Charcot-Leyden crystals were observed in the evacuations, which is of interest in connection with the absence of eosinophilia.

During the early treatment of the case an attempt was made to count all the worms passed; this, however, was abandoned after two weeks, as the number had then run up to 407, and the supply seemed inexhaustible in spite of weekly administrations of thymol in two doses of 30 grains, separated by an interval of two hours and followed by a purgative.

The ova were looked for each week, and gradually decreased, but up to the time of the patient's death, from cerebral hemorrhage, two months after admission, never wholly disappeared.

The general condition improved, but there was always fever, the temperature ranging between 99° and 100°. This continued fever is contrary to the rule.

The last blood count was on August 3, the day before his death, and showed a marked improvement. Reds, 3,033,000 (60 per cent. of normal); whites, 3200; hæmoglobin, 52 per cent.

The appetite was fairly good, the weight had increased, and there seemed to be every reason to hope for a favorable outcome, when, upon August 4, the patient fell unconscious with the symptoms of cerebral hemorrhage, which shortly proved fatal. A similar fate to that of one of Sandwith's cases.

One point which seems worthy of note is the remarkable resistance to treatment shown by the worms. In spite of weekly administrations of thymol in large doses for a period of two months, there were still a few parasites left.

The autopsy was performed by Dr. Nichols.

Necropsy. Body fat and well nourished, but pale. Genitals infantile. The small intestine was of large calibre and had thick walls, especially the duodenum and jejunum. There were no perceptible induration, cicatricial markings, or contractions of the intestinal walls. The valvulæ conniventes were very prominent, and along their bases were rows of very minute points of congestion or hemorrhage; aside from these doubtful points, no distinct hemorrhagic areas, obvious macroscopically, were present. The mucous coat of the bowel was lined with viscid chyme or mucus in which were embedded some uncinaria which were in relatively small numbers. Twenty-nine of the worms were found distributed indifferently along the entire small intestine. A few others may have escaped notice. One of the worms was found with its head attached to the mucosa of the ileum.

The descending colon was studded with small round gray points, apparently enlarged solitary follicles. The mesenteric glands were enlarged. The liver was much enlarged, weighing 2390 grammes; firm, pale in places. The pancreas was firm and hard. The spleen much enlarged, 17x11x5 cm. in size, weighing 410 grammes; congested, fairly firm, and mottled.

The kidney of horseshoe variety, the two organs being united at the lower end; much enlarged, the whole weighing 455 grammes. There was one ureter on each side, both passing down in front of the conjoined kidney. The bladder was distended with urine. The pericardial sac contained a noticeable amount of serum. The heart was much enlarged, weighing 467 grammes. The wall of the left ventricle thickened, the cavity nearly empty, the right containing soft, post-mortem clot. Valves normal. Foramen ovale closed. The left lung much contracted and retracted back into the thoracic cavity, with extensive pleural adhesions easily broken up; weight 273 grammes. Right lung normal, weight 273 grammes.

The brain showed a general subdural hemorrhage over the left cerebrum and a subdural hemorrhage on the right side over the upper portion of the fissure of Rolando. On the right side there was softening of the cerebral substance about the upper end of the fissure of Rolando, giving rise to hemorrhage, the extravasation of blood extending outward under the dura. In the left cerebrum there was similar softening in the occipital lobes with hemorrhage extravasating under the dura. The ventricles contained no blood.

Cultures from liver and spleen were negative.

The only points wherein the pathological findings differed macroscopically from those usually observed in these cases were in the enlargement of the liver and spleen and in the scarcity of ecchymotic spots in the wall of the small intestine. The latter is readily accounted for by the fact that the parasites had nearly all been destroyed before the patient's death.

The microscopical observations, also made by Dr. J. B. Nichols, were as follows:

Specimens removed from various points in duodenum, jejunum, and ileum showed no material alterations. The subepithelial connective tissue of the muscosa was thickened, studded with lymphoid cells. The glandular epithelium was shrunken and apparently degenerated, but this was probably a post-mortem change. The epithelium cells of the glands of Brunner appeared enlarged and pale.

The colon showed enlargement of the solitary follicles with erosion of the overlying mucosa.

The mesenteric glands presented a normal appearance, the enlargement noted macroscopically being due to general hypertrophy of the gland structures. The spleen presented a normal appearance, without congestion or extravasation of blood; its enlargement consisting in a general hypertrophy. The heart-muscle was normal. The kidney showed congestion, bloodvessels and capillaries being filled with blood-cells. The parenchyma was practically normal, no fatty or amyloid change being observed. *The section from the liver was lost.* The only abnormalities noted were the tendencies to hyperplasia of the lymphatic structures.

LAPAROTOMY FOR PERFORATION IN TYPHOID FEVER.

REPORT OF SIX CASES.

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The report is based upon the six cases of laparotomy for intestinal perforation during the course of typhoid fever, occurring in Lakeside Hospital since its opening in January, 1898. These were performed in 1899 and 1900 during the writer's service as resident surgeon, and, with the exception of the last case, came under his immediate observation and care. The inclusion of this last case in the series is perhaps of doubtful propriety, since the patient entered the hospital as a case of general peritonitis and was treated as such, the case containing very little of interest with reference to the surgical treatment of typhoid perforation. For the purpose of completeness, however, it has been added.

It is due to the courtesy of the visiting staffs of Lakeside Hospital that the present report has been made possible, and to these gentlemen the writer wishes to acknowledge his obligation.